EPIGENETIC BIOPOLITICS: RACE, POPULATION, AND ENVIRONMENTAL HEALTH

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Note: This is a first draft of a journal article. Especially at this stage, I welcome comments on all aspects, including target journals (I'm currently thinking something like BioSocieties, but I'm not convinced that is the best place for it).

Introduction

Reading the US government's current warning and advice regarding fish consumption, one would have little hint that race might be important in this environmental health matter. Issued jointly by the EPA and FDA, the advice aims to protect fetuses and young children from the harm that even low doses of methylmercury can cause to brain development (US FDA and US EPA 2004). Because fish is the main source of exposure to methylmercury, this advisory tells women of childbearing age and children which fish they should not eat at all and it provides meal-based advice for fish in general (e.g. eat no more than two meals a week). In this seemingly race-neutral approach, the "vulnerable population" consists of young children and all women who are or who might get pregnant—which elsewhere I have identified as the always already pregnant woman (Mansfield 2012a, 2012b). All people in this vulnerable group can be equally protected by managing the number of meals of fish they eat.

There are, however, hints that race is afoot in these advisories. The pamphlet version of the EPA/FDA advisory—produced for the public—features a photo of a visibly "Asian" pregnant women and child. Further, in addition to English this pamphlet is available in Cambodian, Chinese, Hmong, Korean, Vietnamese, Spanish, and Portuguese. One could interpret this simply as a race-neutral policy that needs to be communicated to all, so that including these other languages and the image of an Asian woman and child is simply an act of racial inclusion in what otherwise remains race-neutral. However, it does

seem from the image and languages that the advisories are especially concerned about people from East and Southeast Asia. Might the "vulnerable populations" being targeted for these advisories not be so race-neutral? And what might be the effects of such advisories—as well as the environmental contamination they are supposed to address—if there are racial disparities at work? More broadly, is the targeting of particular populations for protection from environmental contamination via behavior modification a form of racialized biopolitics? The advisories are quite biopolitical in that they aim to "make life live," and more specifically in that they are a project of securing the population through harnessing the activities of individuals in a subpopulation in order to maximize the present and future health of the population generally (Foucault 1990, 2003). This paper investigates race dimensions of this environmental biopolitics, not only in terms of disparities among already given racial groups but in terms of the process of racialization, understood as the process of race formation (McWhorter 2009; Omi and Winant 1994).

Mercury contamination in fish is just one of many controversies about environmental contaminants and human health bursting forth today. New chemicals such as BPA and PBDEs regularly make the headlines, becoming household words and everyday worries alongside decades-old concern about metals such as mercury and lead, persistent organic pollutants such as PCBs and DDT, and nuclear radiation. Toxicologists, epidemiologists, industry members, regulators, activists, consumers, and parents try to make sense of conflicting evidence regarding the harms of all these chemicals and what—if anything—to do about them. Not only are there new chemicals, but we now have new fears about the effects of our toxic environments. Where once acute toxicity and cancer were the primary concerns, new ideas such as "endocrine disruption" (affecting wildlife and humans) are also gaining traction so that now we are also worried about the broader effects of contaminants on our biochemical bodies: not just deadly cancer but subtle reproductive and neurological effects (such as those caused by methylmercury) are increasingly at the forefront of our fears (Krimsky and Goldman 2002; Langston

2010). With subtle effects came another new twist, which is that the emphasis is no longer simply on *how much* of a chemical we are exposed to, but the *timing* of that exposure: for some chemicals, being exposed to even minute doses *in utero* can have measurable effects where the same exposure in adults has little or no effect. It is all this that animates the fish consumption advisories, which primarily aim to protect fetal neurodevelopment from environmental methylmercury by altering the dietary habits of the always already pregnant woman, who herself would not be harmed by low dose exposures.

The next section of the paper demonstrates that chemicals such as methylmercury, their effects, and our knowledge of them are part of a transformation in our understanding of life, biology, and human health—and hence in our efforts to protect life and health. The section also shows that questions of race are central to this transformation in biopolitics. The following section provides information about methylmercury as an epigenetic hazard, outlining how it has been problematized over the past half century. The next two sections then provide evidence regarding the role of race in attempts by US environmental and health agencies to manage methylmercury as a threat to fetal neurodevelopment. Agency actions appear contradictory: at different times identifying racial disparities in methylmercury exposure via differences in fish consumption, ignoring race but thereby normalizing white diets, and being racially inclusive through carefully communicated consumption advisories.

My argument is that through all these attempts to understand and manage methylmercury as a threat to biosecurity, pregnancy becomes a key site of biopolitical race formation. Because of racial variation in fish consumption, it is women of color who are more impacted by supposedly race-neutral advisories: it is they who are told to change their diets. Further, by normalizing the white diet, this biopolitics of fetal neurodevelopment problematizes methylmercury in new ways. The focus is no longer contamination itself, but instead the abnormal diets of women of color, who must choose to become white to protect themselves and their offspring. Finally, to the extent that they fail to make the right

choice, this leads to bodily differences between people of "different" races. That is, race is the material effect of this epigenetic biopolitics of fetal neurodevelopment.

Race in epigenetic biopolitics

Xenobiotic chemicals and our responses to them are changing how we think about bodies and environments. On the one hand, people consider these human-made toxins as assaults on our given natures. No longer ontologically separate from the environment, we are now open to it in ways that are seen as an existential threat given that they not only kill us outright, but change how we function hormonally, reproductively and neurologically. On the other hand, our chemical fears are part of a broader transformation about how we think about the relationship between "nature" and "nurture." No longer is the "natural" (as the biological) something that is given, but is rather something that is quite changeable.

Whereas existing scholarship has focused on mutability of biology as the result of innovations at the forefront of biotechnology and biomedicine that make it possible to intervene in life as never before (Rose 2007; Rabinow and Rose 2006), I argue that knowledge about pollution and the biological effects of chemicals is leading to similar—and yet quite different—notions about biology and life. Of particular interest are new scientific understandings of bodies that no longer see them as genetically determined, but as "epigenetically" influenced, particularly but not exclusively during fetal development (Heijmans et al. 2009). That is, genes do not act alone, but always in concert with a variety of mechanisms that affect the expression of those genes. Here I use the term "epigenetics" somewhat broadly to refer to cellular processes that influence the things that genes are also thought to influence, such as neurodevelopment. Crucially, the field of *environmental* epigenetics is showing that those cellular processes are themselves influenced both by individual factors of stress, nutrition, and behavior, and by exposure to xenobiotic chemicals—all of which have their greatest effects on developing fetuses (Bollati and Baccarelli 2010).

What makes environmental epigenetics quite different from biomedical intervention, however, is that this form of mutability is rarely in ways of our choosing. These environmental health concerns are precisely about the accidental and indiscriminate movement of molecules—of PCB, of PBDE, of BPA, of methylmercury—through environments and bodies, and how these molecules become biological in their actions: the much-hyped molecularization of life is simultaneously an environmentalization of the molecule.

Epigenetic biopolitics, then, refers to a wide-ranging change in how life—and nature, bodies, biology, and environments—is grasped and understood, but also taken-hold of and governed. To get at this, the question animating this paper is, "*what role does race play in epigenetic biopolitics of the 21st century*?" That is, I ask about the role of race in this changing understanding and governance of biological life. Biopolitics, of course, is the name Foucault gave to the new apparatus of power he saw emerging in the 19th century: an apparatus directed at populations, rather than individuals per se, the aim of which is not to make-die but to make-live (Foucault 1990, 2003, 2007). While biopolitics in this sense seems to be aimed at securing the life of "all" (humans as a *species*), in fact race has been inseparable from biopolitics. "Making live" has always entailed dividing populations into those who live and those who are a threat, i.e. those who must die so that others may live, and race is the key technology for dividing populations in this way (Foucault 2003; McWhorter 2009, 2011; Puar 2007; Stoler 1995). "If sexuality names the strategy of biopower that forces certain people to 'live,' then race serves as the arrangement of power that allows certain people to 'die'" (Stone 2011, 40). Race, in this sense, is difference attached to the body: differences not just of morphology (e.g. skin color) but of reproduction and sexuality, intelligence (or "feeblemindedness"), and morality, all viewed as themselves biologically determined and as threats to the future of the biologically constituted "human race" (McWhorter 2009).

What, then, happens to race if biology is no longer something pre-determined but is always the outcome of processes that are at once molecular and environmental, natural and social? And if race has been at the center of biopolitics, to what extent might epigenetic biopolitics be a technology of racial formation (Omi and Winant 1994)? From the perspective of biomedicalization, the answer has been that race is much less significant now than in the past. Focusing on advances in genomics, many scholars have noted that race simultaneously disappears (genetic mapping reveals that humans are mostly similar, and there are no genes for race) but then reappears, as genetic differences among individuals and populations are then reinscribed as "race" (Koenig, Lee, and Richardson 2008). Rose and Rabinow leave this question of genetic differences open, while arguing that race is becoming much less salient because the target of biopolitics has changed. Biopolitics, they claim, is no longer targeted to management of populations and their "gross characteristics such as intelligence or personality" but instead to individualized intervention operating "according to logics of vitality" (Rabinow and Rose 2006, 208, 211; Rose 2007). The molecularization of life, according to them, brings not only the mutability of biology, but a biopolitical switch from normalization to optimization. It thereby renders moot traditional notions of race, which were premised on differences in kind and used to sort populations. Yet what the Foucauldian race scholars have shown is that race was never an inherent quality—to be undermined by advances in genomics—but that race is a process through which populations are divided by reference to differences attached to the body. This argues against the easy elision of normalization and race by molecularization. Instead, their scholarship urges us to inquire as to how race might contribute to knowledge of epigenetic life, and how epigenetic life reconstitutes—rather than erases—race, and with what effects.

What I will show is that, far from making race meaningless, epigenetic biopolitics marks a transformation and even intensification of racialization. To the extent that biology is mutable, then evidence that childbearing women of color fail to properly manage their individual and collective bodies

doubly proves that race exists, and exists on the body. First, through their improper management, they show that they are indeed different: incapable of being the rational, liberal subject, always implicitly racialized as white. Secondly, this incapacity remakes their bodies and those of their children: difference becomes quite material, in the form of altered neurodevelopment. This is the process of making race a biophysical difference from a white norm, written not in practices but in the structure of the brain, the working of the body, and the remaking of the reproductive system. Whereas race may have started as a fiction—a social construction—through epigenetic biopolitics it is made quite material, not just in phenomenological embodiment, but in the molecular-environmental development of individuals. Far from disappearing, population security comes to the fore. It proceeds through the marking of individuals as unworthy failures and as biophysically different: they become the threat (to themselves) that must be eliminated—if not killed then disciplined—in order to make life live. Further, what seems different about this sorting process is that it starts from the premise that all people *can* be proper liberal subjects. This is about how the racialized process of *inclusion* is also the moment of discipline (Mansfield 2012b). The biopolitical moment is also the disciplinary moment, which is also the moment of producing racial formations—differences that are attached and attributed to the body. In this epigenetic biopolitics, in which the aim is to affect cellular processes of the developing fetus, it is the always already pregnant woman who is racialized and who, through her actions, produces embodied race.

Methylmercury as an epigenetic hazard

Methylmercury as an environmental hazard has several salient features that make it paradigmatic of epigenetic biopolitics. First, it is impossible to classify methylmercury contamination as a human or natural phenomenon. Mercury is a naturally occurring element that today is released into the atmosphere through coal burning and waste incineration; it is then widely dispersed and deposited on land and water. Mercury in aquatic systems is transformed by bacteria into its highly toxic form of methylmercury, which then bioaccumulates along food chains so that it is most concentrated in

predatory fish; these fish are the main source of methylmercury exposure for humans. Methylmercury in fish is at once a ubiquitous "environmental" problem with biological consequences for people, and an illustration of how both "the environment" and thus "the body" are neither natural nor social.

Second, study of methylmercury contamination since the 1950s has contributed directly to a transformation in toxicological understanding of how chemicals affect human bodies, with a new focus on fetal development as a critical window of toxicity (for summary, see Grandjean et al. 2010). The neuro-toxic effects of methylmercury—creating symptoms such as sensory disturbances and tunnel vision—were first noted in the mid-19th century, though they were then largely forgotten and methylmercury subsequently was used as a fungicides for seeds (used especially in the Green Revolution). The acute toxicity of methylmercury for adults was rediscovered (and dose-response relationships described) following a series of tragic poisoning events from the 1950s-1970s, in which people either directly consumed treated seed grain (e.g. Iraq in the 1950s and early 1970s), or consumed fish from water bodies polluted with industrial discharge (e.g. Japan in the 1950s and 1960s). In what proved to be a great challenge to toxicological models, these poisoning events also provided early evidence that methylmercury is not only an acute poison, but affects children born by women who are exposed during pregnancy. In these events, children who were exposed only *in utero* showed measurable neurological effects—sometimes years after the exposure occurred. Crucially, their mothers may not have shown any signs of poisoning: doses that were safe for adult women were not safe for their offspring. Thus, it is not just the dose that matters, but the timing. The EPA uses methylmercury as its example when explaining this shift in emphasis more generally (US EPA 2010b):

> Almost 500 years ago Paracelsus (1493-1541) wrote: "Dosis facit venenum" or "the dose makes the poison." The relationship between dose and response (health effect) is still one of the most fundamental concepts of toxicology - or is it? For pollutants that act as developmental toxicants, the

> same dose that may pose little or no risk to an adult can cause drastic effects in a developing fetus or a child. Methyl mercury is but one example of a chemical that is much more toxic early in life.

Third, the neurological effects at issue with regard to fetal development are quite subtle, so that our fears are not about pathology as illness but about *abnormality*. Following the findings from the poisoning events, much of the research from the mid-1990s to today has aimed to identify dose-response relationships for fetal exposures, particularly through epidemiological studies of the effect of women's fish consumption on children's neurodevelopment. The aim is to identify the most sensitive "endpoint" and identify the dose below which there is no effect (i.e. the threshold). These endpoints are no longer symptoms of toxicity, but rather subtle signs of neurological abnormality that can be identified only using sensitive neurodevelopmental testing (e.g. neurological, sensory, and intelligence tests). Even then, the presence of these abnormalities (and the dose and timing of exposure that might cause them) remains highly contested, as the two largest studies have had different results. The study in the Faroe Islands found that mercury exposure was correlated with reduced scores on several neurological and intelligence tests, while the study in the Seychelles found no such correlations (e.g. Davidson et al. 2011; Grandjean et al. 2003). While these contradictory results created quite a stir during the late 1990s, it seems now that the lack of effect found by the Seychelles study is likely due to the "confounding" effects of the positive health benefits of eating fish (most likely the presence of healthful omega 3 fatty acids). This itself has caused debate, even among the studies' investigators: does this indicate that mercury in fish is worse than thought (effects of mercury are *hidden* by the benefits of fish) (Budtz-Jorgensen, Grandjean, and Weihe 2007) or is better (such effects are *balanced out* by the benefits of fish) (Strain et al. 2008)?

Fourth, as these epidemiological studies and debates suggest, while the biophysical effects of methylmercury are on individual bodies, the problem is grasped as a question of population health. In

this context, risk analysis is now the organizing framework for gathering and making sense of disparate information about methylmercury, information derived from numerous studies in toxicology and toxicokinetics, epidemiology, exposures, risk communication, and so on (Mansfield 2012a). It is important to note here that "hazard" and "risk" are not conceptually or materially the same. Hazards are dangers; e.g. methylmercury is hazardous to fetal neurodevelopment. Risk is a form of governance, an attempt to calculate and manage that which is fundamentally unknowable, i.e. the future, population-level effect of such hazards. Populations are the basic unit in risk analysis because risk is necessarily a relative, rather than absolute measure, derived by measuring distributions in populations. For methylmercury, not only is the concern about subtle effects to fetuses (rather than acute effects in adults), but individual effect will be quite small; thus, the explicit concern of risk analysis is population-level effects, that is, the aggregate effects of small reductions in, for example, intelligence (e.g. Cohen et al. 2005).

In sum, methylmercury in fish is problematized as the effect of low-dose exposures of environmentally ubiquitous methylmercury during fetal development, and the endpoints of concern are subtle abnormalities in neurodevelopment, influencing physical responses, developmental trajectories, and mental abilities commonly grouped as "intelligence." As subtle, fetal effects, the problem can only be discerned at the population level, and becomes one of (future) population security. Risk analysis is then the primary technique for governing this environmental health concern. Even as threshold doses are calculated at the individual level (the amount of methylmercury an individual can safely ingest) this is simply the way of grasping hold of the population; the individual threshold dose is the way to manage population exposures to and outcomes from this socio-environmental molecule that shapes—in still surprising ways—the course of human development.

Risk and vulnerable populations: race-neutral and racialized

To understand how this new biopolitics of xenobiotic chemicals draws on and transforms racial formations, the rest of this paper analyzes risk analysis of methylmercury in the US, focusing on the efforts of US agencies, primarily since the 1980s, to understand and manage this environmental health issue. Risk analysis breaks "risk" down into a series of discrete questions about the population effects of chemical hazards and effective means for managing population exposures and outcomes. It is commonly noted that risk analysis does not seek to *eliminate* either hazardous chemicals or exposure to them, but rather to *manage* exposures so as to reduce negative effects to an "acceptable" level (Harrison 2011). In other words, risk is a biopolitical technology for "letting things happen" and managing effects at the population level (Foucault 2007). Elsewhere I have analyzed the risk-benefit approach to methylmercury in fish (dominant since about 2000), which is driven by the notion that the harms of mercury need to be "balanced" against the benefits from eating fish (Mansfield 2012a). This paper primarily addresses the period prior to this, when the focus was whether mercury poses a population risk and what to do about that—and finds that race is a central, though not always explicit, theme of this process.

My discussion here and in the rest of this paper is based on analysis of the extensive documentation by US government agencies of their efforts to understand and manage the health effects of methylmercury. The primary agencies with responsibility for methylmercury are the Food and Drug Administration (FDA) and Environmental Protection Agency (EPA). The agencies are evaluated and advised by panels of scientists convened by the Institute of Medicine (IOM) and National Research Council (NRC), and the EPA, in particular, also works with state and tribal regulatory bodies.

The FDA, which regulates commercial fish only, first released "action limits" for mercury in commercial fish in the 1970s, and then released consumer advisories regarding consumption of these fish starting in the early 1990s; their archive provides information about how those advisories were

developed over time. The EPA, which regulates "recreational" fish, published its first risk assessment for methylmercury in 1985, and then during the Clinton administration produced thousands of pages of analysis and regulation of mercury as a health hazard. This includes the agency's massive, 7-volume 1997 Mercury Study Report to Congress (hereafter "1997 Report"), which was conducted to fulfill a requirement of the 1990 Clean Air Act (US EPA 1997). In a 2000 publication, the NRC provided scientific review of the 1997 Report (National Research Council 2000). Using the 1997 Report and the 2000 NRC review, in January 2001 (in the very last days of the Clinton administration) the EPA produced a non-binding water quality criterion for methylmercury (US EPA 2001); the guidance to states about using the criterion was released in 2010 (under the Obama administration) (US EPA 2010a). In the 1990s the EPA also produced a 4-volume National Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories (hereafter "National Guidance") for states and tribes, still current as of this writing (US EPA 2012a). Also available are the proceedings from the National Forum on Contaminants in Fish, convened by the EPA seven times since 1999 as a venue for discussion among people from states, tribes, and other interested groups (US EPA 2012b).

This section and the next use this body of evidence to identify the seemingly very different ways that race is taken into account, calculated, and produced by these agencies in the different stages of risk analysis. This section focuses on the risk assessment stages: toxicity assessment (hazard identification and dose-response relationships) and risk characterization (including exposure assessment). The toxicity assessment of methylmercury appears largely race-neutral, as it focuses on identifying biological links between methylmercury and negative health outcomes. What emerge are different vulnerabilities associated with timing (fetal development) and gender (childbearing women), but not with race. Risk characterization, on the other hand, is not at all race-neutral. Instead, what emerge are racial disparities regarding which people are potentially exposed to methylmercury via their diets.

Race-neutral toxicity assessment

Toxicity assessment comprises three main questions. First, is there credible toxicological and epidemiological evidence that humans are affected by the chemical in question, that is, is there a hazard? Second, if so, what is the relationship between different exposures and their particular health outcomes, or "endpoints." The central goal is to identify the specific individual dose below which adverse effects are not expected; this dose is referred to by a variety of terms, but is conceived of as a "threshold." The highly complicated process of identifying a numerical threshold requires a series of decisions (for example, which critical study, endpoint, toxicokinetic model, statistical model, and approach to uncertainty). Third, is risk equally distributed across the population, or is there a biologically "sensitive" subpopulation which is more physiologically susceptible? The goal is to calculate the dose that will protect not just the "general" population but the "vulnerable" population as well. Thus, the EPA describes the threshold—which they call a "reference dose"—this way (US EPA 1997, vol. 1, p. O-2):

> The reference dose (RfD) is an amount of methylmercury, which when ingested daily over a lifetime is anticipated to be without adverse effects to humans, including sensitive subpopulations. At the RfD or below, exposures are expected to be safe. The risk following exposures above the RfD is uncertain, but risk increases as exposures to methylmercury increase.

By the 1980s, the answer to the first question was clearly that methylmercury is a human health hazard, and both the EPA and FDA used risk analysis to identify threshold doses based on the most sensitive effects in adults (i.e. paresthesia); the EPA's threshold was more protective (i.e. lower) than the FDA's.

By this time, however, it was becoming clear that fetuses were more biologically susceptible to the effects of methylmercury than were adults. This new focus on timing is incorporated in terms of the "sensitive population," which—answering the third question—for methylmercury became not only all

fetuses but all women of childbearing age. As the EPA put it, "because the developing fetus may be the most sensitive to the effects from methylmercury, women of child-bearing age are regarded as the population of greatest interest" (US EPA 1997, vol. 1, p. O-3). In 1995 the EPA released a new, much lower threshold dose, based on a 1985 study of fetal effects from the Iraqi poisoning event in the 1970s. This was justified in excruciating detail in the 1997 Report, which was itself evaluated in the 2000 NRC report analyzing emerging evidence from the conflicting epidemiological studies mentioned earlier. The EPA's 2001 water quality criterion incorporated both its 1997 Report and the NRC analysis, and presented a "new" EPA threshold dose—which numerically was exactly the same as the 1995 threshold: 0.1 micrograms of methylmercury per kilogram body weight per day. This is the threshold level that the EPA continues to use as of this writing (IRIS 2012).

In all calculations of the threshold since the 1980s, the focus is on vulnerable, or sensitive, subpopulations, which were defined biologically as all fetuses and women of childbearing age. Evidence suggests few biological differences among people that would make some more susceptible physiologically—there are no biological differences among "races"—so that the dose-response relationship is defined to be the same among all people of the same age and sex. That is, the population is differentiated based on timing (pregnancy and childhood), but not race. Hence, toxicity assessments from the 1990s on are race-neutral, in that they found no correlation between socially defined race and physiological susceptibility.

Racially differentiated risk characterization and exposure assessment

Following toxicity assessment is a process of risk characterization, which asks about under what (if any) real-world scenarios this hazard becomes a real problem. Once a threshold dose has been calculated, under what circumstances are people actually exposed at levels above that threshold? Exposure assessment becomes important at this stage: does the "general population" or a subset

thereof eat enough fish high in methylmercury to be exposed at levels above the threshold? I will show that that the agencies approach this question in different ways. The FDA focuses on the average consumer, thereby normalizing average consumption. The EPA, in contrast, focuses on variability in consumption, identifying who eats a lot of fish and why. They find that variability is racial: people who eat a lot of fish are disproportionately people of color. Seen in this light, the FDA's race-neutral focus on the average is also racial, in that it posits "white" diets as "normal."

Normalization at the FDA

In 1986, the FDA published a risk assessment for methylmercury that yielded an "acceptable daily intake" of 30 micrograms per day (in contrast to EPA's current RfD, which yields seven mcg/day for a 70 kg person) (Tollefson and Cordle 1986). Their exposure assessment was based on dietary consumption surveys from the early 1970s, commissioned by the Tuna Research Foundation. Purportedly designed to be representative of the US population, the survey included geographic variables (e.g. census region) but none for race or ethnicity. Analyzing the statistical distribution of fish consumption from these data, the FDA concluded that "the probability of a systematic exposure to substantial intakes of methylmercury in fish and shellfish by the average consumer is low" (1986, 206), and the notion of the "average" consumer is repeated several times. At the same time, their analysis identified that a small percentage of the US population eats a large amount of fish. Combining consumption information with mercury levels in specific species, the FDA concluded that even at the 95th to 98th percentile (i.e. the 2-5% of consumers with very high fish consumption), these people would still be exposed at levels less than 30 mcg/day. Thus, using statistical means of population management, they conclude that "U.S. fish consumption data do not indicate any cause for concern of methylmercury poisoning for the average American" (1986, 208). Therefore, the average diet is used as the measure of exposure, and no new regulation is necessary to protect the "average American."

A 1991 IOM panel was highly critical of the FDA's 1986 risk assessment (Institute of Medicine 1991). Featured prominently in the critique was the FDA's focus on the average consumer. The panel recommended that the FDA instead use sophisticated statistical means to determine how many people might approach or exceed the threshold. Despite this, the FDA continued to fuse the "average American" and the "normal diet" through the 1990s and into the 2000s. In its 1990's advisories, the FDA suggests limiting consumption of some species, but states that "consumption advice is unnecessary for the top 10 seafood species" (a notion that itself implies normality) (Foulke 1994). This is because methylmercury levels in those species are low and because "few people" eat enough to have significant exposure. The advisory even goes so far as to say that it is not the "normal pattern of consumption" that puts people at risk, but only "when people eat fad diets" such as eating only a particular species of fish. In the 2001 update, the FDA specifically included information not just on which fish to avoid but which fish to eat, and in its rationale it that stated that this is a "balanced diet" (US FDA 2001a, 2001b). The 2004 advisory similarly urges women to eat a "well-balanced diet that includes a variety of fish and shellfish," where balanced means eating some fish but not too much, and not eating a lot of any one type of fish (US FDA and US EPA 2004). These later approaches to exposure assessment also have been criticized, for example by several commentators at a 2002 FDA advisory committee meeting, who, in different ways, noted that the "one-size-fits-all" approach ignores that some people do eat a lot of fish, and for a variety of reasons (US FDA 2002). These commentators all argued that such people—not just normal, average consumers—deserve protection from methylmercury.

Thus, since the 1980s the FDA has relied on the statistical average for determining regulatory action, and in so doing it has equated the average and the "normal," often using the terms interchangeably. Protection, for the FDA, is entirely for the "average American" and not for anyone who exceeds the norms with abnormal, unbalanced, "faddish" diets. The course of action, then, is to have people change their diets: to become normal. While clearly dealing with the normal/abnormal divide that is the focus

of much Foucauldian scholarship of race, the FDA's approach is more obviously racialized when we understand just who is at the statistical tails: who is it that has an abnormal, faddish diet and needs to be normalized?

By the 1980s there was awareness that "high-end" fish consumers are not equally distributed across the population but instead are concentrated especially among groups of people who rely on fish for subsistence: fish as an inexpensive source of healthful food, sometimes a food that also has important cultural value. For example, the 1991 IOM report clarified that "many 'recreational' anglers may actually be subsistence anglers who fish as an important means of supplementing their diet, and they may share their catch with family members and friends" (Institute of Medicine 1991, 246), and stated in its summary that "consumers of recreational or subsistence fishery products" are at risk from environmental contaminants (1991, 2). They hint at racial dimensions of this when further subdivide the category of recreational into "sport, subsistence, and tribal fishing" (1991, 222). Citing studies from the 1980s, they note that these fishers may be much more highly exposed than average, for example because they may fish in one (highly contaminated) area and they may consume large amounts of fish from these areas (1991, 245). It is true that "recreational" fish are beyond the remit of the FDA. Yet, because it is the cumulative levels across all exposure sources that matter, such consumption would need to be taken into account (as "background") when calculating whether fish is a real hazard.

The EPA documents racial disparities in exposure

In the early 1990s, the EPA raised the problem of focusing on the "average" and instead aimed to identify so-called high-end consumers; in so doing, the agency brought race to the forefront—at least for awhile. A 1992 report on "Consumption surveys for fish and shellfish" justifies the need for such surveys by stating that "the use of an 'average' consumption rate…may not accurately reflect the local consumption rate in a particular subpopulation" (US EPA 1992, 1). Citing studies from the 1980s, it goes

on to state that “certain subpopulations, based on race, ethnic origin, age, sex, income, and residence, did consume more fish than other groups… Minorities from cities, rural Native Americans, and the elderly also caught and consumed more fish” (US EPA 1992, 3). Identifying and discussing these disparities remained a strong focus of the EPA for the next decade. Thus, the 1997 Report states in its executive summary, “The typical US consumer eating fish from restaurants and grocery stores is not in danger of consuming harmful levels of methylmercury from fish…[but] analysis of dietary surveys led the US EPA to conclude that between 1 and 3 percent of women of child-bearing age… eat sufficient amounts of fish to be at risk from methylmercury exposure” (1997, vol. 1, pp. O:2-O:3). Whereas the FDA discounted the small percentage of women who ate a lot of fish, for the EPA they are the focus: “high-end consumers of local fish are clearly a subpopulation of concern” (1997, vol. 6, p. 5:4). Biopolitical questions regarding how to measure and calculate disparities in fish consumption then figure centrally in the exposure assessment volume of the 1997 Report (and subsequently the risk characterization volume), where a central question is that of diet: who are “high-end” fish consumers and just how much fish are they eating? Over the course of ninety pages, the exposure volume slices population fish consumption in multiple ways, including by age, gender, and race (1997, vol. 4).

To do this, the EPA relies on two basic sorts of evidence. One is published studies of fish consumption and mercury exposure of specific groups. Such studies, over time, have identified both rural and urban subsistence fishers as high-end fish consumers, primarily (though not exclusively) for economic reasons. These subsistence fishers disproportionately include African Americans, Asian Americans, and many immigrants, especially those from Southeast Asia and the Caribbean where fish are commonly eaten. The Report gives Native Americans special attention because some tribes (particularly in Alaska, along the Pacific coast, and around the Great Lakes) “traditionally consume high quantities of fish and fish products” (1997, vol. 4, p. 4:34). It is further noted that, for some of these tribes, fish is important for cultural as well as economic reasons (1997, vol. 4, p. 4:44). The other form of

evidence is analysis of data from national dietary surveys such as the National Health and Nutrition Examination Survey (I do not address here the myriad problems with these surveys). Using statistical analysis to identify high-end consumers (i.e. those at the tail of the distribution, such as the 95th or 99th percentile), the 1997 Report shows that, at the population level, people of color eat more fish more frequently than do "white" people, and subsequently are expected to have higher mercury exposures. For example, while two to three percent of adults ate fish six or more times a week (1997, vol. 4, p. 4:17), this included two percent of "whites", 3.3 percent of "blacks", and nine percent of "other," a problematic catchall category that includes, *inter alia*, Asian and Pacific Islanders, Native Americans, and non-Mexican Hispanics (1997, vol. 4, p. 4:18). Among people who eat fish, the top five percent of "white" people are eating 243 grams per day, compared to 302 grams for "black" people, 292 grams for "Asian and Pacific Islander" people, and 327 grams for "other" people (1997, vol. 4. p. 4:24). The 1997 Report estimates mercury exposure across one month, finding that exposure exceeded the EPA reference dose for nine percent of "white" fish eaters, 12.7 percent of "black" fish eaters, and 16.6 percent of "other" fish eaters (1997, vol. 6, p. 6:30)—and notes that it is underestimating exposures for anyone who eats fish that are more contaminated than average. In sum, the 1997 Report makes it clear that there are people in the US that eat enough fish to be of concern given the current reference dose for methylmercury, and that these are disproportionately "ethnically and racially defined subpopulations" (1997, vol. 6, p. 6:29).

The EPA's National Guidance to states and tribes wanting to develop consumption advisories also emphasizes the distinct issues facing Native Americans, particularly those involved in subsistence fishing. Here, the EPA is explicit that their approach is distinct from that of the FDA; they say that states and tribes should follow EPA methodology because "the underlying assumptions used in the FDA methodology were never intended to be protective of recreational, tribal, ethnic, and subsistence fishers who typically consume larger quantities of fish than the general population and often harvest the

fish and shellfish they consume from the same local waterbodies repeatedly over many years" (US EPA 2000, 1:11). They go on to say that the EPA recognizes "that Native American subsistence fishers are a unique subsistence fisher population that needs to be considered separately. For Native American subsistence fishers, eating fish is not simply a dietary choice that can be completely eliminated if chemical contamination reaches unacceptable levels; rather, eating fish as in integral part of their lifestyle and culture" (US EPA 2000, 1:12).

As these documents demonstrate, the EPA was cognizant not only of variation in fish consumption, but that high-end consumption is not equally distributed in the population. Rather, there are specific, racialized groups that eat more fish, and therefore have greater exposure to methylmercury. By the beginning of the 2000s, however, the explicit focus on race starts to fall away, even in discussion of exposure. Once the variability had been identified, there was a move to subsume race within a seemingly race-neutral rhetoric on hazards to high-end consumers, whoever those consumers may be. There were hints of this even in the 1997 Report, in which summary sections such as the executive summary overview (presumably written last and read most) lacked any explicit mention of race, instead referring to high-end consumers as a group and differences by age and gender. This is more evident in the 2000 NRC review of the 1997 Report, which emphasizes that "individuals with high MeHg exposures from frequent fish consumption might have little or no margin of safety (i.e., exposure of high-end consumers are close to those with observable adverse effects)" (2000, 9). Yet the NRC just briefly summarizes who these individuals are, and it does so with no reference to race. Methylmercury exposure is mentioned a problem for "subsistence fish eaters" with no discussion of who these are (2000, 15), and variability is explained in terms of "individual characteristics" and differences in the "characteristic amounts and types of fish consumed in different regions of the United States," with one reference to "specific ethnic and cultural subgroups" (2000, 38, 39). The dominance of a race-neutral construction of high-end consumers is also evident in the EPA's 2001 Water Quality Criterion for the

Protection of Human Health: Methylmercury, which is a major outcome of the agency's study of this issue in the 1990s. That document describes how the EPA calculated a level of methylmercury in fish tissue to be protective of human health, focusing on high-end consumers (defined as those above the 90th percentile) in three groups: the general public, childbearing women, and children (US EPA 2001).

Thus, it seems that racial disparities disappear just as they appear. Just as in the 1997 Report—in which attention to race disappears in the overview, in favor of identifying the one to three percent of women who may be at risk—racial disparities in these later documents disappear in the technical (and biopolitical) language of 90th percentile fish consumers. So doing ignores and thereby hides that while it may be one to three percent of women in the US who are at risk of having their children exposed to a developmental neurotoxin, it is much more than three percent of black women, Asian American women, and Native American women, though exactly how many is never made clear.

My point here has been twofold. For one, I want to show that, indeed, variation in exposure to methylmercury is racially differentiated, with people of color in the US being much more highly exposed. Therefore, methylmercury in fish can be seen as akin to classic environmental justice issues of racially disproportionate exposure to environmental toxins. The second point, following from this, is to contrast how the EPA dealt with the question of variability with the normalizing approach taken by the FDA. The FDA, in taking the supposedly race-neutral approach of focusing on the "typical" American diet, ignores variability and multiplicity. But evidence of racial disparity makes it clear that the FDA's approach would have a highly racialized pattern of negative impact. That is, identifying the "high-end" consumer highlights that "typical" is itself raced and classed: it is the average middle-class white diet (which itself might be a statistical artifact, describing no real people). Fish-eating becomes racialized: non-white diets are non-normative, so that the "average" (50th percentile) consumer is racialized (implicitly) as white—and white diets are normative. The seemingly race-neutral approach of focusing on the typical assumes that all individuals have equal chances to be at a particular spot on the distribution when, as the EPA

shows, people of color, especially if poor, are more likely to be at the high-end of the distribution than are white people. Policies that fail to address disparities further exacerbate disparities (Omi and Winant 1994), leaving people of color to be exposed to potentially harmful environmental contaminants.

Risk management: advisories as inclusive and racializing

Following toxicity assessment and risk characterization is a process of risk management and communication. These are the stages in which risk analysts develop strategies for managing exposures—for keeping individuals under the threshold dose and managing population outcomes. This section asks how the agencies' strategies of risk management address the question of racial disparities in exposure. It is here that the seemingly race-neutral fish consumption advisories come in, as the dominant strategy for managing population exposure today. Advisories are targeted to all women who are or could become pregnant, with no mention of race, and they offer this target population specific advice about how many meals of what sorts of fish they should be eating. Risk communication then aims to be racially inclusive, so that everyone, regardless of race, learns and acts on the advisories.

I argue here that regardless of how race-neutral a meal-based advisory appears or how inclusively it is communicated, advisories are a racializing technology for comprehending and managing methylmercury as a public health issue; that is, they enact a racial epigenetic biopolitics. There are at least two dimensions to this. The first is that the advisories themselves have racially disparate effects, because disproportionately people of color are the ones who have to change their diets. The second is that advisories not only have disparate outcomes, but they recast the problem as one of racial difference. Risk analysis transforms the problem from one of methylmercury to once of racially marked, abnormal diets, and both the EPA and FDA together enact a racialized program of normalization that produces race as an idea and creates real biological differences among previously racially marked groups.

Advisories as racist dispossession

It might seem that advisories are a positive way of dealing with racial disparities in methylmercury exposure. That is, rather than identifying and targeting the "typical" consumer they acknowledge variability in exposure. Indeed, the EPA at times has treated advisories as an environmental justice issue. This was explicit in the risk management volume of the National Guidance for developing fish advisories, released in the mid-1990s but still current today: "environmental justice is particularly relevant to the work discussed in this document because contaminated fish may be consumed in greater quantities by minorities and low-income populations" (US EPA 1996, 1:16). And as this document noted, many racial justice advocates, tribes, and community groups have called for *more and better communicated consumption advisories*, on the grounds that not letting people know about toxins in their fish is a form of racist harm. One cited example was from Chicago, where community leaders saw the failure to post advisories at fishing sites at which many black people fish for food as a sign of indifference to their well-being (US EPA 1996, 2:9, 3:9). In this context, the focus of both regulatory agencies (EPA, state, tribal) and many community groups is to find ways to better communicate advisories, as a means of racial inclusiveness (e.g. US EPA 1996, 2:18; National Risk Communication Conference 2001).

However, another response from tribes and environmental justice organizations has been to see the advisories as themselves a form of racist harm. By their nature advisories are targeted to the "atypical": the people who eat a lot of fish, especially in contaminated areas. As the EPA itself demonstrated, these are primarily Native Americans and people of color, often poor, both rural and urban. It is these atypical people of color who have to change—including people for whom subsistence fishing is central part of their livelihood strategy and/or cultural identity. In this sense, focusing on making advisories inclusive and well-communicated fails to address the key problem, which is that of pollution. Many tribal representatives told the EPA that advisories are problematic because they pass responsibility for pollution onto disadvantaged communities while letting polluters of the hook (US EPA 1996). The EPA,

under pressure from such groups, acknowledged this issue in its guidance on developing fish advisories. "Many individuals consulted from community and tribal groups requested information regarding environmental remediation and pollution prevention be included in this volume. These groups frequently expressed the sentiment that the ultimate goal should be to improve environmental quality so that fish advisories are no longer necessary. This has been the EPA's goal since its inception" (US EPA 1996, 1:19). However, the agency said this in a document on developing advisories, and as it was moving forward with advisories as the main technique for managing contaminated fish.

Tribes and environmental justice advocates, however, continued to challenge advisories as the solution and to demand a focus on pollution control and remediation. In the late 1990s and early 2000s, many of these challenges came at the National Forum on Contaminants in Fish. A representative from the Confederated Tribes of the Umatilla Indian Reservation, in Oregon, put it this way (Harris 2001):

> I demand the freedom to consume any and all parts of all the foods that my elders have taught me are the center of our cultural and spiritual lives without fearing for my life or the lives of my children…I feel that advisories may be useful, but only as an unfortunate interim necessity. Responding to fish contamination is not just a communication problem…It is not a problem of communicating risk across a cultural divide… We need to see the EPA setting goals, taking action and standing firm to make things safe again… Ultimately we need to clean up the fish and the river, and we need to do it before anymore cultural knowledge is lost.

Or, as an anonymous speaker put it, "tribal input on brochures; I don't want a brochure. I want the EPA to turn the responsibility back to the polluters in our country and not put it on us" (National Risk Communication Conference 2001, III-31). Members of the Umatilla Indian Reservation argued that the

loss of fish (through contamination and advisories) "is analogous to the loss of the buffalo to the Plains Indians" (National Risk Communication Conference 2001, F-6). As the 2000s continued, however, mention of pollution remediation is absent in formal discussion of consumption advisories, and issues of racially diverse impacts goes missing. Even at the National Forum, by the mid-2000s the focus was narrowed to technical issues of sampling, biomonitoring, communication, and—even more—the idea of "balancing" risks and benefits, a problematic that makes harms much more difficult to understand (see Mansfield 2012a).

In this way, both environmental contamination and advisories designed to solve the problems of contamination run through the grooves of existing racial inequality, thereby deepening these grooves. While appearing on their face race-neutral and inclusive, advisories most impact people of color in the United States. By increasing hardship and eliminating an important part of people's diet and cultural practice, the dominant solution to the problem of methylmercury is a form of dispossession of important resources. Advisories thereby worsen inequalities in areas such as food access, cultural integrity, and health outcomes.

Race as an effect of advisories

Yet advisories do more than deepen the grooves of existing inequality. Rather, advisories at once treat the problem *as* one of racial difference, and do so in ways that are materially *productive of* that difference. If the regulatory focus is advisories, then the target of governance is non-normative diets. Here, both the FDA and EPA with their very different approaches are quite similar. Both agencies, by governing through advisories, normalize the "white" diet and pathologize racialized diets. Once advisories with disparate racial effects become the answer, then the problem to be governed is no longer methylmercury but instead the abnormal diets of these racially different people. At issue is directing women of color to make the proper individual dietary choices. This is evident in the ways risk

analysis has recast the problem of methylmercury exposure in the past two decades. Reading documents from the early 1990s, the problem seems to be the effect of pollution on children born to women who are exposed, which focuses attention on the pollution and treats women as a population of concern. By the 2000s, once the shift to advisories as a technique was complete, the problem is recast as effects on children who were exposed as fetuses by their mothers. Certainly this shifts the blame to *mothers*, who no longer are themselves exposed but who now expose their children via their dietary choices (Mansfield 2012b). But evidence about racially differentiated patterns of fish consumption suggests it is more than this. The problem is that *women of color* expose their children through their non-normative diets, in which they eat too much fish, and from the wrong sources.

In this scenario, it is not that women and girls of color are disproportionately exposed because a major food source is contaminated, but that these racialized childbearing women are exposing their children because they make bad dietary choices. The women are the problem, and the solution is not to prevent or cleanup pollution, but to demand that women of color adopt white diets. If they "fail" to do so for whatever reason, they fail both as deserving citizens and as biochemical bodies, proving their difference from the white norm twice over. They fail to protect their babies, their futures, their population from toxic chemicals that cause biophysical changes, giving birth to children more likely to have sensory issues and problems with learning—and it is their own fault. As the member of the Umatilla Indian Reservation quoted earlier put it, "I have been told there may be people in regulatory positions who think that people who don't comply with advisories are dumb, or uneducated, or deserve to get sick if there is a way to avoid fish. I know that I will be blamed for not complying with an advisory" (Harris 2001, III-36). Since "abnormal" diets produce "abnormal" brains—with developmental and cognitive changes—this is, indeed, the production of biological difference following pre-defined racial lines.

Seen this way, the meal-based fish consumption advisories appear not as a race-neutral tool for protecting the general population, but as a racializing technology that divides the population by normalizing whiteness. Risk analysis has recast the problem and solution in ways that makes race more rather than less salient, and that could even lead to new biophysical differences among people of purportedly different "races." A woman's abnormal, racialized diet is written on her child's brain. Suddenly, racial differences in intelligence—long one of the key axes of racialization (McWhorter 2009)—become real. The reality of such racial differences would be especially apparent, as "truth," when measured as populations: methylmercury might only affect a small slice of, say, black people, yet still be measurable on a population basis, so that it might be possible to find significant differences in intelligence among "blacks" and "whites." Inclusive risk communication, in which the advisories are communicated to multiple populations, while in one sense protective, is also a way to enroll people in this racial project of normalizing whiteness and attaching difference to the non-normative body.

Conclusions

This paper has argued that risk analysis of methylmercury exposure is a biopolitical strategy that differentiates the population and attaches those differences to the biological body, which is itself considered behavioral and environmental. This epigenetic biopolitics, far from eliminating race by eliminating notions of differences in kind written in the genes, has intensified it by relying on notions about how bodies are created through the supposedly race-neutral behaviors of liberal subjects. In contrast with the FDA, which actively ignored variability, the EPA started with a project of inclusion in which it identified who is really being harmed by methylmercury pollution. The project was to identify variation across racial lines, with everyone part of the population needing protection from harm to fetal neurodevelopment. But then, to ensure biosecurity they insist that people of color change what they do: eat differently. This serves not only as a means to dispossess disadvantaged groups of resources and to remove responsibility from polluters. It also changes what the problem is: not pollution, but the non-

normative diets "chosen" by women of color. The problem is not the toxic environment but the ways individuals do or do not manage the movement of that environment through their bodies and cells.

The moment that advisories become the answer, the liberal subject is at once assumed and denied. Biology may be mutable—life epigenetic—but *because of this* it becomes not just individual opportunity but individual responsibility to manage that. The ability to choose is taken for granted. The problem is non-white women, who do not properly protect themselves and their offspring. If they fail in their choices—fail as liberal subjects—they cause harm to themselves and their children. Not only does this cause racial differences to become biologically real, but it also "proves" that race was real all along. The fact of difference now (e.g. lower IQ) cannot be explained without difference before. That is, women of color prove that they never were liberal subjects. This is abnormality, attached to the body; this is the sorting mechanism, necessary to less optimistic accounts of the politics of "make live." It is because biology is not given but made that race becomes more important. In a world in which biological outcomes are made (they are our own responsibility), then differences in outcomes show that we are in fact different, and it is that difference that constitutes race. Individualized optimization is not a post-racial promise, but rather a gendered and racialized demand. Race is the outcome that proves itself.

Nor does this sort of individual optimization require that we choose between inclusion (as liberal subjects, part of the population to be protected) or exclusion (as exception, that part of life dangerous to itself and so sacrificed). Here we see that—through race—we have both, intimately tied, and in the same body (individual and collective). Not only in the name of protecting "others," but in the name of protecting themselves, racialized subjects are at once assumed to be full liberal subjects capable of securing the population through their behavior, and are shown to be incapable of such: to be a threat. The always already pregnant black woman, Asian woman, Native woman becomes the racialized other, who either becomes white or proves her difference twice over: as the one who is given the chance to be the liberal subject and fails, and, due to this failure, as biologically different: producing offspring who

bear the biophysical signs of her failures, becoming-other. Difference is attached to the epigenetic body, and this difference is abnormality: this is race.

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